We claim:

1. A concatemerized double-stranded oligonucleotide molecule comprising at least two copies of a nucleotide sequence comprising a sequence or sequences that act as transcription factor decoys.

2. A transcription factor decoy comprising concatemerized double-stranded oligonucleotide molecule at least two end-to-end repeated copies of a nucleotide sequence comprising a sequence or sequences that act as transcription factor decoys.

3. A combinatorial transcription factor decoy comprising concatemerized double-stranded oligonucleotide molecule at least two end-to-end nucleotide sequence comprising two different sequences that act as transcription factor decoys for 2 or more transcription factors.

4. The transcription factor decoy of claim 1, further comprising at least one tissue-specific promoter.

5. The transcription factor decoy of claim 1, wherein the decoy is capable of blocking signaling and gene expression associated with pathogenesis.

6. The transcription factor decoy of claim 1, wherein the decoys are NF-kB-specific.

7. The transcription factor decoy of claim 1, wherein the transcription factor is selected from NF-kB, AP-1, ATF2, ATF3, and SP1.

8. A method of delivering transcription factor decoys *in vitro* or *in vivo*, in isolated cells or intact animals, comprising comprising concatemerized double-stranded oligonucleotide molecule at least two end-to-end repeated copies of a nucleotide sequence comprising a sequence or sequences that act as transcription factor decoys.

9. The method of claim 8 wherein the transcription factor decoys block transcription factors implicated in a disease, response to surgery and/or trauma, developmental defects, aging, toxic exposure.

10. The method of claim 8 wherein the treatment is for the treatment of one or more of the diseases selected from the group consisting of myocardial ischemia/reperfusion and myocardial infarction, heart failure and hypertrophy, cardioprotection, stroke, neuroprotection, sepsis, arthritis, asthma, heritable inflammatory disorders, cancer, heritable immune dysfunctions, inflammatory processes, whether caused by disease or injury or infection, oxidative stress to any organ whether caused by disease, surgery or injury.

11. A method for treatment of NF-κB-associated diseases which comprises administering to an animal an effective amount of a polynucleotide NF-κB chromosomal binding site decoy which antagonizes NF-κB-mediated transcription of a gene located downstream of a NF-κB binding site wherein the polynucleotide comprises one or more copy of the oligonucleotide decoy.

12. The method according to claim 11 wherein the NF-κB-associated disease is selected from the group consisting of; an ischemic disease, an inflammatory disease, and an autoimmune disease.

13. The method according to claim 11 wherein the NF-κB-associated disease is an ischemic disease.

14. The method according to claim 11 wherein the NF-κB-associated disease is selected from the group consisting of; a reperfusion disorder in ischemic disease, aggravation of a prognosis of an organ transplantation, aggravation of a prognosis of an organ surgery, a post-PTCA restinosis.

15. The method according to claim 11 wherein the NF-κB-associated disease is selected from the group consisting of; a reperfusion disorder in ischemic heart disease, aggravation of a prognosis of a heart transplantation, aggravation of a prognosis of a heart surgery, and post PTCA restinosis.

16. The method according to claim 11 wherein the NF-κB-associated disease is selected from the group consisting of; a cancer metastasis a cancer invasion, and cachexia.

17. A method of treating a nuclear factor κB-dependent disease selected from the group consisting of immunological disorders, septic shock, transplant rejection, radiation damage, reperfusion injuries after ischemia, arteriosclerosis and neurodegenerative diseases, comprising administering to a mammal in need of such treatment an effective amount of an oligonucleotide decoy.

18. The method of claim 17 wherein the oligonucleotide decoy is delivered by a polymeric vector.

19. The method of claim 17 wherein the nuclear factor-κB-dependent disease is an immunological disorder.

20. The method of claim 17 wherein the nuclear factor-κB-dependent disease is septic shock.

21. The method of claim 17 wherein the nuclear factor-κB-dependent disease is transplant rejection.

22. The method of claim 17 wherein the nuclear factor-κB-dependent disease is radiation damage.

23. The method of claim 17 wherein the nuclear factor-κB-dependent disease is reperfusion injury after ischemia.

24. The method of claim 17 wherein the nuclear factor-κB-dependent disease arteriosclerosis.

25. The method of claim 11 wherein the nuclear factor-κB-dependent disease is a neurodegenerative disease.

26. The method according to claim 11 wherein the administering inhibits cell death and apoptosis in ischemic-reperfused myocardium.

27. The method according to claim 11 wherein the administering inhibits apoptosis in ischemic-reperfused brain, reducing neuronal cell death in stroke.

28. The method according to claim 11 wherein the administering inhibits apoptosis in the failing heart, reducing apoptosis cell death in congestive heart failure and cardiomyopathy.

29. A therapeutic method comprising treating non-aortal procedural vascular trauma comprising administering to a mammal, subjected to the procedural vascular trauma, an effective protective amount of an oligonucleotide decoy, or a pharmaceutically acceptable salt thereof.